Integrative & Personalised Medicine 22

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Viral and bacterial infection in multisystem disease, and reactivation by SARS-CoV-2

INTEGRATIVE AND PERSONALISED MEDICINE CONGRESS 2022

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Agenda

- **Involvement of bacteria and viruses in multisystem disease**
- SARS-CoV-2/COVID-19 and viral reactivation
- Testing
- Checklists and other resources

Multiple stealth infections can underlie the "mysterious diseases" of our time: bacteria …

- Borrelia burgdorferi (3 subspecies: B.b. sensu stricto + B.b. garinii + B.b. afzelii) – causative agent of Lyme Disease
- Borrelia myamotoi
- Bartonella
- Babesia
- Chlamydia pneumoniae
- Chlamydia trachomatis
- Mycoplasma pneumoniae
- Ehrlichia/Anaplasma
- Rickettsia
- Yersinia enterocolitica
- Toxoplasma
- Coxiella burnetti
- Campylobacter jejuni
- Etc.

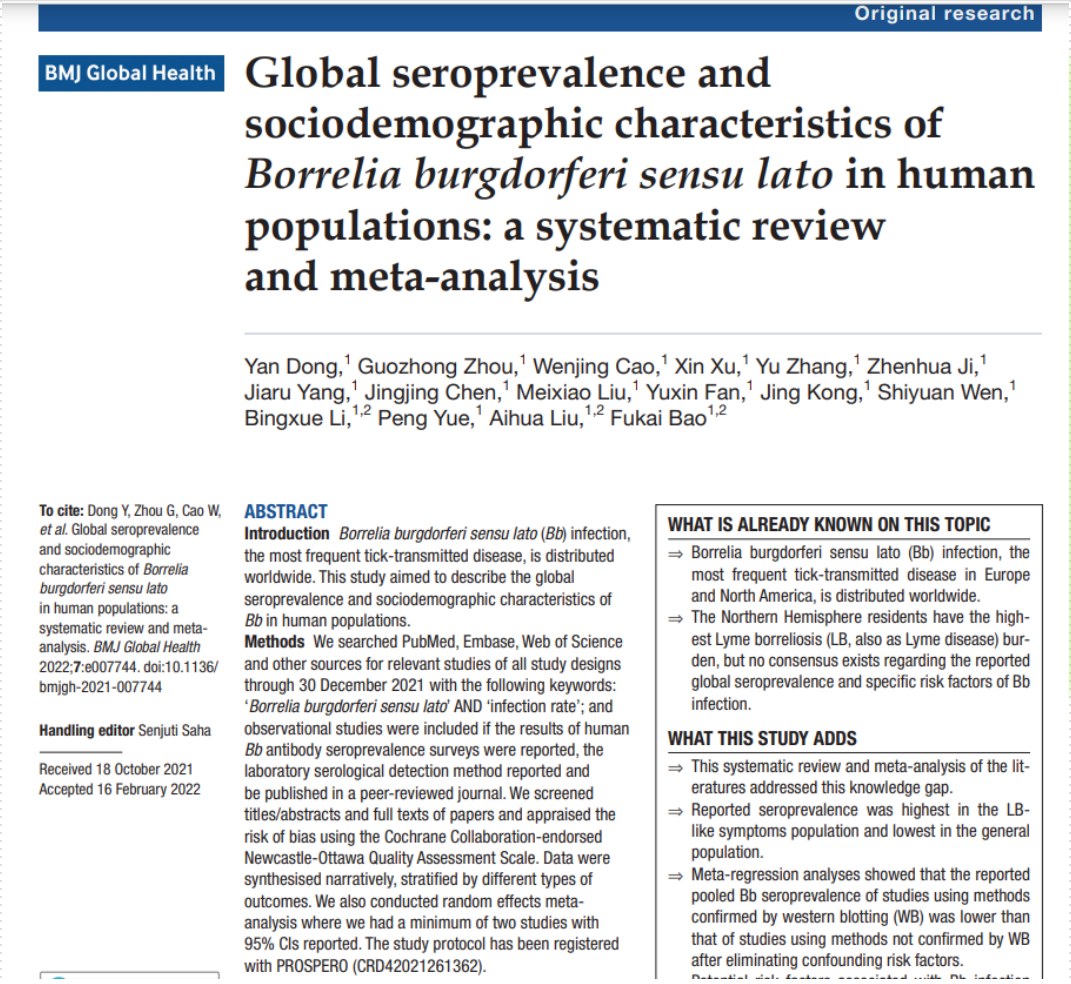
… as well as viruses and other pathogens

- Epstein Barr Virus (EBV)
- Cytomegalovirus (CMV)
- Herpes Simplex Virus 1 / 2
- Varicella Zoster Virus (VZV)
- HHV-6, HHV-7, HHV-8
- Parovirus B19
- Coxsackievirus A & B
- Echovirus
- SARS-CoV-2

- Parasites (e.g. Leishmania, Echinococcus, Taenia solium, Entamoeba histolytica, Toxocara canis, Trichinella spiralis)

- Mycotoxins

Nearly 15% of the world is seropositive for Borrelia according to first global estimate (Feb. 2022)



Original research

BMJ Global Health

Global seroprevalence and sociodemographic characteristics of *Borrelia burgdorferi sensu lato* in human populations: a systematic review and meta-analysis

Yan Dong,[1] Guozhong Zhou,[1] Wenjing Cao,[1] Xin Xu,[1] Yu Zhang,[1] Zhenhua Ji,[1] Jiaru Yang,[1] Jingjing Chen,[1] Meixiao Liu,[1] Yuxin Fan,[1] Jing Kong,[1] Shiyuan Wen,[1] Bingxue Li,[1,2] Peng Yue,[1] Aihua Liu,[1,2] Fukai Bao[1,2]

To cite: Dong Y, Zhou G, Cao W, *et al*. Global seroprevalence and sociodemographic characteristics of *Borrelia burgdorferi sensu lato* in human populations: a systematic review and meta-analysis. *BMJ Global Health* 2022;**7**:e007744. doi:10.1136/bmjgh-2021-007744

Handling editor Senjuti Saha

Received 18 October 2021
Accepted 16 February 2022

ABSTRACT

Introduction *Borrelia burgdorferi sensu lato* (*Bb*) infection, the most frequent tick-transmitted disease, is distributed worldwide. This study aimed to describe the global seroprevalence and sociodemographic characteristics of *Bb* in human populations.

Methods We searched PubMed, Embase, Web of Science and other sources for relevant studies of all study designs through 30 December 2021 with the following keywords: '*Borrelia burgdorferi sensu lato*' AND 'infection rate'; and observational studies were included if the results of human *Bb* antibody seroprevalence surveys were reported, the laboratory serological detection method reported and be published in a peer-reviewed journal. We screened titles/abstracts and full texts of papers and appraised the risk of bias using the Cochrane Collaboration-endorsed Newcastle-Ottawa Quality Assessment Scale. Data were synthesised narratively, stratified by different types of outcomes. We also conducted random effects meta-analysis where we had a minimum of two studies with 95% CIs reported. The study protocol has been registered with PROSPERO (CRD42021261362).

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Borrelia burgdorferi sensu lato (Bb) infection, the most frequent tick-transmitted disease in Europe and North America, is distributed worldwide.

⇒ The Northern Hemisphere residents have the highest Lyme borreliosis (LB, also as Lyme disease) burden, but no consensus exists regarding the reported global seroprevalence and specific risk factors of Bb infection.

WHAT THIS STUDY ADDS

⇒ This systematic review and meta-analysis of the literatures addressed this knowledge gap.

⇒ Reported seroprevalence was highest in the LB-like symptoms population and lowest in the general population.

⇒ Meta-regression analyses showed that the reported pooled Bb seroprevalence of studies using methods confirmed by western blotting (WB) was lower than that of studies using methods not confirmed by WB after eliminating confounding risk factors.

The reported estimated global Bb seroprevalence was 14.5% (95% CI 12.8% to 16.3%), and the top three regions of Bb seroprevalence were Central Europe (20.7%, 95% CI 13.8% to 28.6%), Eastern Asia (15.9%, 95% CI 6.6% to 28.3%) and Western Europe (13.5%, 95% CI 9.5% to 18.0%).

MS and Borrelia – research over 4 decades

medical hypotheses

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< Previous Article February 1988 Volume 25, Issue 2, Pages 89–92 Next Article >

Multiple Sclerosis is a chronic central nervous system infection by a spirochetal agent

Vincent Marshall
Animal Vaccine Laboratory, 255 Elliott Street, Council Bluffs, Iowa 51501 USA

PlumX Metrics

DOI: https://doi.org/10.1016/0306-9877(88)90023-0

Abstract References

Abstract

Multiple Sclerosis (MS) is a chronic central nervous system (CNS) infection similar to Lyme Disease (1) or Neurosyphilis in its latency period, pathogenesis, symptoms, histopathology and chronic CNS involvement. It does not have as yet a fully identified spirochetal etiological agent. Much research and clinical support for this hypothesis was published before 1954 and is based on silver staining of neural lesions, animal isolation of the etiologic agent and the characteristic symptoms and pathogenesis of the disease. If this hypothesis is correct, the disease should be treatable with antibacterial agents that penetrate the CNS (such as high dose antibiotics), diagnosible by specific immunological tests, and preventable by early treatment or by the use of vaccines in high risk populations.

Med Hypotheses. 2005;64(3):438-48.

Chronic Lyme borreliosis at the root of multiple sclerosis--is a cure with antibiotics attainable?

Fritzsche M[1].

Author information

Abstract

Apart from its devastating impact on individuals and their families, multiple sclerosis (MS) creates a huge economic burden for society by mainly afflicting young adults in their most productive years. Although effective strategies for symptom management and disease modifying therapies have evolved, there exists no curative treatment yet. Worldwide, MS prevalence parallels the distribution of the Lyme disease pathogen Borrelia (B.) burgdorferi, and in America and Europe, the birth excesses of those individuals who later in life develop MS exactly mirror the seasonal distributions of Borrelia transmitting Ixodes ticks. In addition to known acute infections, no other disease exhibits equally marked epidemiological clusters by season and locality, nurturing the hope that prevention might ultimately be attainable. As minocycline, tinidazole and hydroxychloroquine are reportedly capable of destroying both the spirochaetal and cystic L-form of B. burgdorferi found in MS brains, there emerges also new hope for those already afflicted. The immunomodulating anti-inflammatory potential of minocycline and hydroxychloroquine may furthermore reduce the Jarisch Herxheimer reaction triggered by decaying Borrelia at treatment initiation. Even in those cases unrelated to B. burgdorferi, minocycline is known for its beneficial effect on several factors considered to be detrimental in MS. Patients receiving a combination of these pharmaceuticals are thus expected to be cured or to have a longer period of remission compared to untreated controls. Although the goal of this rational, cost-effective and potentially curative treatment seems simple enough, the importance of a scientifically sound approach cannot be overemphasised. A randomised, prospective, double blinded trial is necessary in patients from B. burgdorferi endemic areas with established MS and/or Borrelia L-forms in their cerebrospinal fluid, and to yield reasonable significance within due time, the groups must be large enough and preferably taken together in a multi-centre study.

PMID: 15617845 DOI: 10.1016/j.mehy.2004.09.011

[Indexed for MEDLINE]

Dr. Vincent Marshall's exhaustive research from the 80's showed spirochetes on the axons of nerves of MS patient autopsies in Europe.

Source: https://www.ncbi.nlm.nih.gov/pubmed/15617845; http://www.medical-hypotheses.com/article/0306-9877(88)90023-0/abstract

Does EBV underlie MS?: First "compelling evidence of causality" (Science, Jan. 2022)

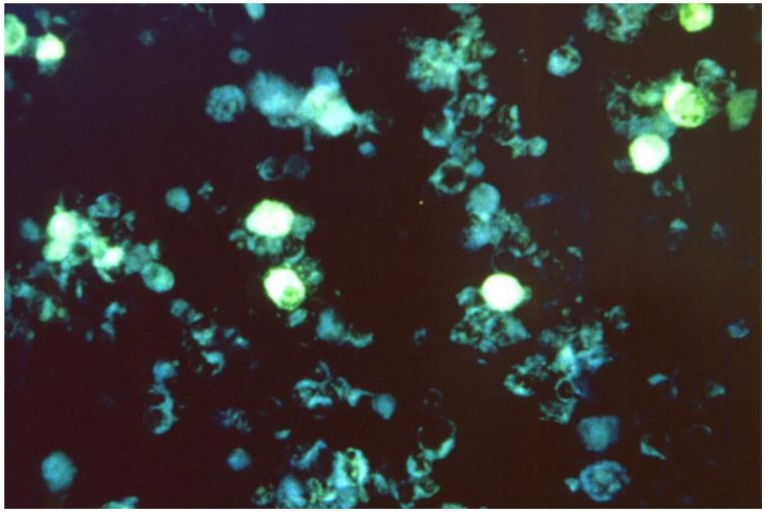
The Harvard Gazette

Cells infected with Epstein-Barr, a common herpes virus that can cause mononucleosis and establishes a latent, lifelong infection of the host.

CDC

HEALTH & MEDICINE

Epstein-Barr virus may be leading cause of MS

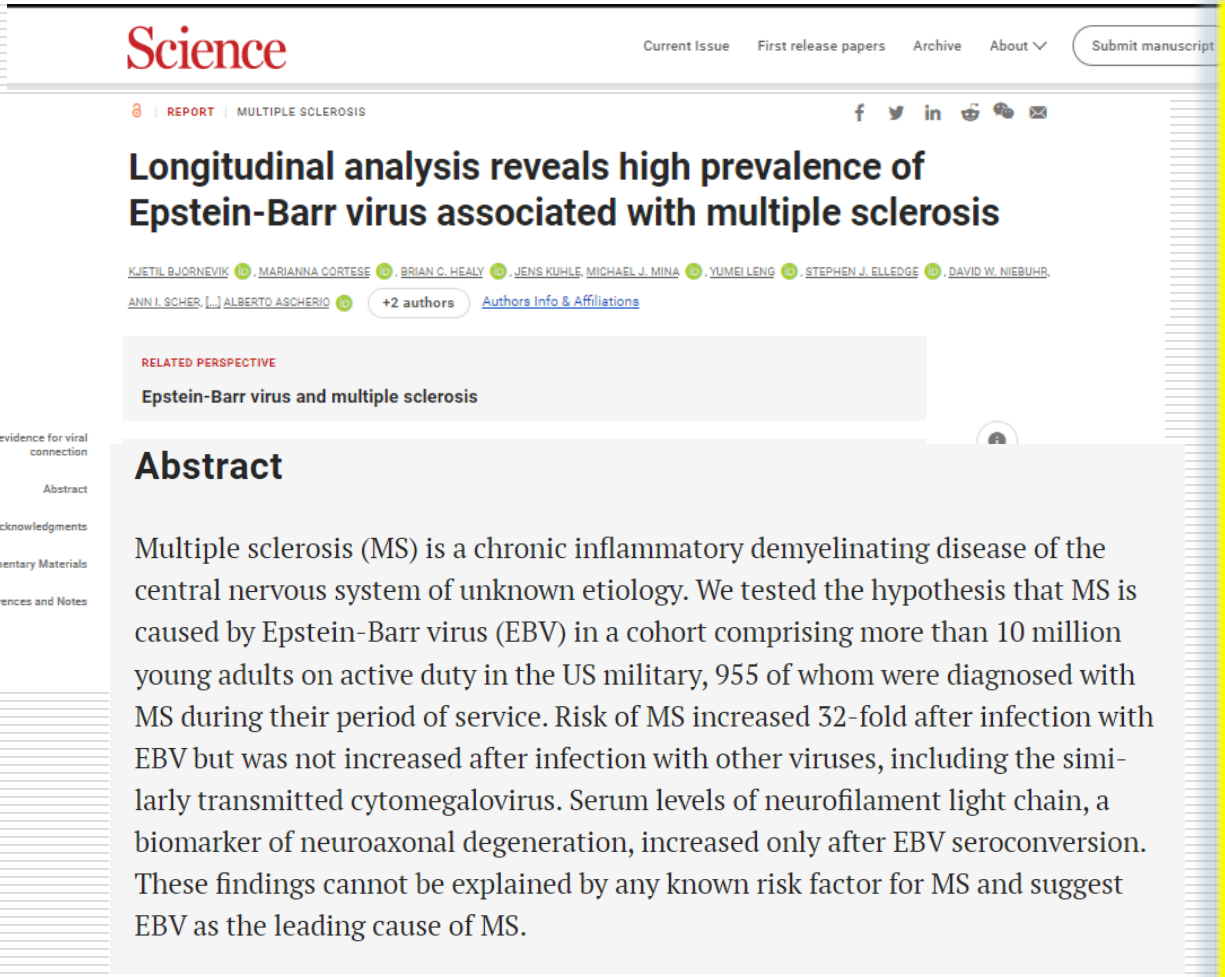


First study to provide 'compelling evidence of causality'

"The hypothesis that EBV causes MS has been investigated by our group and others for several years, but this is the first study providing compelling evidence of causality," said Alberto Ascherio, professor of epidemiology and nutrition at Harvard Chan School and senior author of the study. "This is a big step because it suggests that most MS cases could be prevented by stopping EBV infection, and that targeting EBV could lead to the discovery of a cure for MS."

Source: https://news.harvard.edu/gazette/story/2022/01/epstein-barr-virus-may-be-leading-cause-of-multiple-sclerosis/

The study found that the risk of developing MS increased 32-fold following EBV infection



Science

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REPORT | MULTIPLE SCLEROSIS

Longitudinal analysis reveals high prevalence of Epstein-Barr virus associated with multiple sclerosis

KJETIL BJORNEVIK, MARIANNA CORTESE, BRIAN C. HEALY, JENS KUHLE, MICHAEL J. MINA, YUMEI LENG, STEPHEN J. ELLEDGE, DAVID W. NIEBUHR, ANN I. SCHER, [...] ALBERTO ASCHERIO +2 authors Authors Info & Affiliations

RELATED PERSPECTIVE

Epstein-Barr virus and multiple sclerosis

Stronger evidence for viral connection

Abstract

Acknowledgments

Supplementary Materials

References and Notes

Abstract

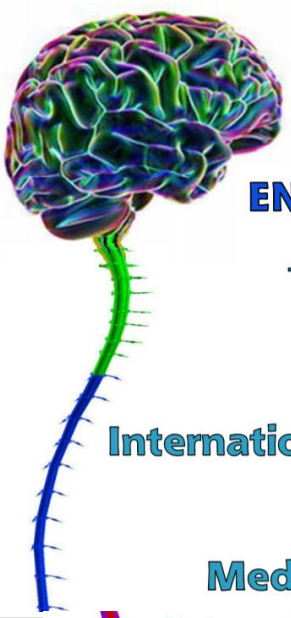
Multiple sclerosis (MS) is a chronic inflammatory demyelinating disease of the central nervous system of unknown etiology. We tested the hypothesis that MS is caused by Epstein-Barr virus (EBV) in a cohort comprising more than 10 million young adults on active duty in the US military, 955 of whom were diagnosed with MS during their period of service. Risk of MS increased 32-fold after infection with EBV but was not increased after infection with other viruses, including the similarly transmitted cytomegalovirus. Serum levels of neurofilament light chain, a biomarker of neuroaxonal degeneration, increased only after EBV seroconversion. These findings cannot be explained by any known risk factor for MS and suggest EBV as the leading cause of MS.

"Using data from more than ten million United States military recruits monitored over a 20-year period, 955 of whom were diagnosed with MS during their service, Kjetil Bjornevik *et al.* tested the hypothesis that MS is caused by EBV. They found that the risk of developing MS in individuals who were EBV-negative increased by 32-fold following EBV infection. "These findings," say the authors, "cannot be explained by any known risk factor and suggest EBV as the leading cause of MS."

Source: https://www.science.org/doi/10.1126/science.abj8222; https://www.eurekalert.org/news-releases/939665

The ME International Consensus Primer recommends considering multiple viruses, Borrelia burgdorferi and other bacteria

M.E.



MYALGIC ENCEPHALOMYELITIS

- Adult & Paediatric:

International Consensus Primer for Medical Practitioners

International Consensus Panel

Editors: Bruce M. Carruthers, MD, CM, FRACP(C)
Marjorie I. van de Sande, B Ed

RION • Causal Factors • Phases

• blood transfusions • anaesthetics • toxic chemicals • heavy metals • severe physical trauma: whiplash/spinal injury/surgery • undue psychological stress[17-23]

Onset Survey
1,000+ patients
75.6%: infection alone or infection + 1 or more factors:
environmental exposure,
physical trauma,
vaccinations,
other stressors
Vernon SD. CFIDS of America

Precipitating Events and Causal Factors: Most patients enjoyed healthy, active lifestyles prior to the onset of ME. Widely dispersed epidemics support an infectious cause. Symptoms at onset are usually consistent with an infectious process.

1. **Infectious agents associated with ME**

Viruses: • Enterovirus[24-26] • Epstein Barr virus (EBV)[27] • Human herpes virus (HHV 6 and 7)[28, 29] • Cytomegalovirus[30] • Parvovirus B19[31]

Bacteria: Chlamydophila pneumonia[32] • Mycoplasma[33] • Coxiella burnettii[27]

It is unclear whether these infectious agents initiated ME or are opportunistic and developed due to an impaired immune system. No one virus has been universally implicated for all patients. A prospective study reported that six months following acute infections of Epstein-Barr virus, Coxiella Burnetii, or Ross River virus, 11% of the patients had CFS.[34] This supports the presence of ME subtypes. Antibody testing for a number of viruses revealed subtype-specific relationships for Epstein Barr virus and enterovirus, two of the most common infectious triggers for ME.[27]

2. **Possible etiological process:** A growing body of evidence suggests that a primary cause of ME is neuropathic viruses that may infect neurological and immune cells and damage the capillaries and micro-arteries in the CNS bed causing diffuse brain injury. The initial infection may cause profound dysregulation of immune system pathways that may become chronic or cause autoimmunity even when the level of the infectious agent is reduced.[35]

Pathogen	*Tests*	*Pathogen*	*Tests*
☐ Enterovirus	RT-PCR, serology, stomach biopsy	☐ mycoplasma	DNA-PCR, serology
☐ EBV, ☐ CMV, ☐ HHV-6	DNA-PCR, serology, antigenemia	☐ Borrelia burgdorferi	DNA-PCR, serology, Western Blot
☐ Clamydia pneumonia	DNA PCR, serology	☐ Parvovirus B19	DNA-PCR, IgG, IgM,
Immune system profiles: ☐ ***↓NK cell function & ↑ cytotoxicity**; ☐ B & T-cell function: ☐ IgG, ☐ IgG subclasses 1-4; ☐ IgA, ☐ IgM (shift from T1 to T2), ☐ cytokine/chemokine profile panel (94% accuracy): IL-8, IL-13, MIP-1β, MCP-1, IL4, ☐ flow cytometry for ↑ lymphocyte activity, ☐ ↑ 37 kDa 2-5A RNase L immunoassay – defect/ratio & bioactivity, ☐ food sensitivity panel, ☐ chemical sensitivities, ☐ stool for WCB - D-lactic acid bacteria balance, ova & parasites, ☐ autoimmune profile, **Intestinal dysbiosis:** ☐ IgA & IgM for intestinal aerobic bacteria in serum, ☐↑ leukocyte elastase activity in PBMCs, ☐ IgG food intolerance test, ☐ toxoplasmosis			
Neurological & static testing: ☐ ***SPECT scan with contrast** - ↓ cortical/cerebellar region cerebral blood flow (rCBF) in the frontal, parietal, temporal and occipital & brain stem regions - *more brain involvement indicates increased illness severity*, ☐ **MRI of brain –** *(increased T2-weighted images in high white matter tracts & loss of GM volume) & rule out MS*, ☐ **MRI of spine** *(dynamic disc bulges/herniation , stenosis)*, ☐ **sleep study** *(↓ stage 4 sleep, sleep pattern & rule out treatable sleep dysfunctions – upper airway resistance syndrome, sleep apnea, etc.)*			

Source: https://doctorswith.me/international-consensus-primer-for-medical-practitioners/

Important to consider Borrelia in the differential diagnosis of rheumatoid arthritis

Clinical and Vaccine Immunology
CVI Article | Journal Info. | Authors | Reviewers | Permissions | Journals.ASM.org

Clin Vaccine Immunol. 2007 Nov; 14(11): 1437–1441. PMCID: PMC2168181
Published online 2007 Sep 19. doi: 10.1128/CVI.00151-07

Serum Reactivity against *Borrelia burgdorferi* OspA in Patients with Rheumatoid Arthritis

Yu-Fan Hsieh,[1] Han-Wen Liu,[1] Tsai-Ching Hsu,[1] James C.-C. Wei,[2] Chien-Ming Shih,[3] Peter J. Krause,[4] and Gregory J. Tsay[1,2,*]

Author information ▶ Article notes ▶ Copyright and License information ▶

ABSTRACT Go to:

Lyme arthritis and rheumatoid arthritis share common clinical features and synovial histology. It is unclear whether they also share similar pathogenesis. Previous studies have shown that the severity and duration of Lyme arthritis correlate directly with serum concentrations of antibody against outer surface protein A (OspA) of the causative pathogen *Borrelia burgdorferi*. We tested the sera of 68 subjects with rheumatoid arthritis, 147 subjects with other autoimmune diseases, and 44 healthy subjects who had never had Lyme

Fibromyalgia and Epstein Barr Virus/CMV

Journal of Neurology and Neuroscience

Evaluation of Antiviral Antibodies against Epstein-Barr Virus and Neurotransmitters in Patients with Fibromyalgia

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Citation: Reshkova V, Kalinova D, Milanov I. Evaluation of Antiviral Antibodies against Epstein-Barr Virus and Neurotransmitte Patients with Fibromyalgia. J Neurol Neurosci. 2016, 6:3. doi: 10.21767/2171-6625.100035

Received Date: August 25, 2015; **Accepted Date:** November 10, 2015; **Published Date:** November 14, 2015

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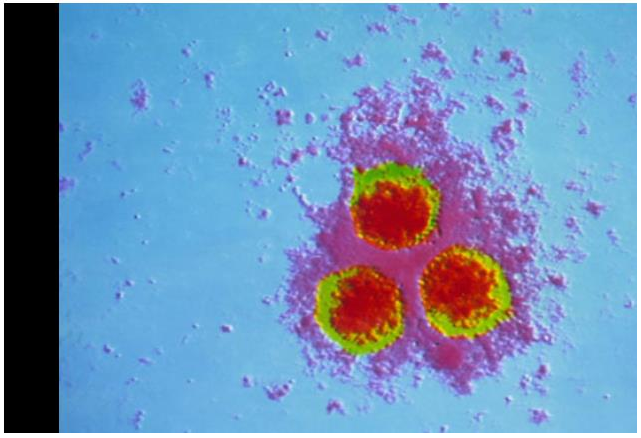
Abstract

Fibromyalgia (FM) is characterized by chronic widespread pain lasting for a minimum of three months, and pain at mechanical pressure in at least 11 of the 18 tender points. The cause of fibromyalgia is unknown. Several hypotheses have been developed including "central sensitization". This theory proposes that fibromyalgia patients have a lower threshold for pain because of increased reactivity of painsensitive neurons in the spinal cord or brain. Some researchers supposed that different

"The obtained results revealed that high EBV IgG concentrations in the serum of patients with FM correlated with pain intensity and associated clinical symptoms. This is consistent with the fact that FM is connected to the immune response to certain infectious agents (e.g. EBV, CMV)."

Fibromyalgia and Varicella Zoster Virus (VZV)

Varicella-zoster virus



Varicella Zoster Virus (VZV) - a highly contagious virus that spreads from person-to-person by coughing or sneezing, or through direct contact with the characteristic skin lesions it causes or fluids from blisters on an infected person.

Curr Top Microbiol Immunol. Author manuscript; available in PMC 2011 Apr 14.
PMCID: PMC3076592
Published in final edited form as:
NIHMSID: NIHMS259279
Curr Top Microbiol Immunol. 2010; 342: 243–253.
doi: 10.1007/82_2009_3

Neurological Disease Produced by Varicella Zoster Virus Reactivation Without Rash

Don Gilden, Randall J. Cohrs, Ravi Mahalingam, and Maria A. Nagel

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Abstract

Go to:

Reactivation of varicella zoster virus (VZV) from latently infected human ganglia usually produces herpes zoster (shingles), characterized by dermatomal distribution pain and rash. Zoster is often followed by chronic pain (postherpetic neuralgia or PHN) as well as meningitis or meningoencephalitis, cerebellitts, isolated cranial nerve palsies that produce ophthalmoplegia or the Ramsay Hunt syndrome, multiple cranial nerve palsies (polyneuritis cranialis), vasculopathy. myelopathy, and various inflammatory disorders of the eye. Importantly, VZV reactivation can produce chronic radicular pain without rash (zoster sine herpete), as well as all the neurological disorders listed above without rash. The protean neurological and ocular disorders produced by VZV in the absence of rash are a challenge to the practising clinician. The

"Zoster is often followed by chronic pain (postherpetic neuralgia)"

Source: https://www.clinicaladvisor.com/varicella-zoster-virus/slideshow/377/

M.E. | Fibromyalgia

Association of Parovirus B19 with ME/CFS as well as fibromyalgia

sciendo

PROCEEDINGS OF THE LATVIAN ACADEMY OF SCIENCES. Section B,
Vol. 73 (2019), No. 5 (722), pp. 411–418.

DOI: 10.2478/prolas-2019-0065

ASSOCIATION OF HUMAN PARVOVIRUS B19 INFECTION WITH DEVELOPMENT AND CLINICAL COURSE OF MYALGIC ENCEPHALOMYELITIS / CHRONIC FATIGUE SYNDROME

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Contributed by Modra Murovska

Our aim was to estimate the presence of B19V infection markers, the level of cytokines and time period since the appearance of infection in association with ME/CFS clinical symptoms. In 200 ME/CFS patients and 104 control group individuals the presence of B19V-specific IgG/IgM class antibodies, B19V NS1 gene sequence, mRNA expression, viral load and level of cytokines were determined. B19V-specific IgG-antibodies were found in 70% of ME/CFS patients and 67.4% of controls, IgM-antibodies in 8% of patients and in none of controls, B19V genomic sequences in 29% of patients and 3.8% of controls. 58.6% of positive patients had active and 41.4% had latent/persistent B19V infection. B19V NS1 gene expression was detected in 43% of patients. B19V load varied from < 0.2 copies to median 38.2 copies/μg of DNA. According to the antibody pattern, 36% of patients had a recent, and 43% had sustained B19V infection. Patients with the B19V genomic sequence and NS1 specific antibodies significantly more often had lymphadenopathy and multi-joint pain. Onset of the symptoms corresponded to time of appearance of B19V infection. IL-10 and TNF- levels were higher in patients with elevated B19V load. B19V genome 1 was identified in Latvian ME/CFS patients. The results indicated that at least in some cases

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Human Parvovirus B19 Infection Status in Patients with Myalgic Encephalomyelitis/Chronic Fatigue Syndrome and Fibromyalgia

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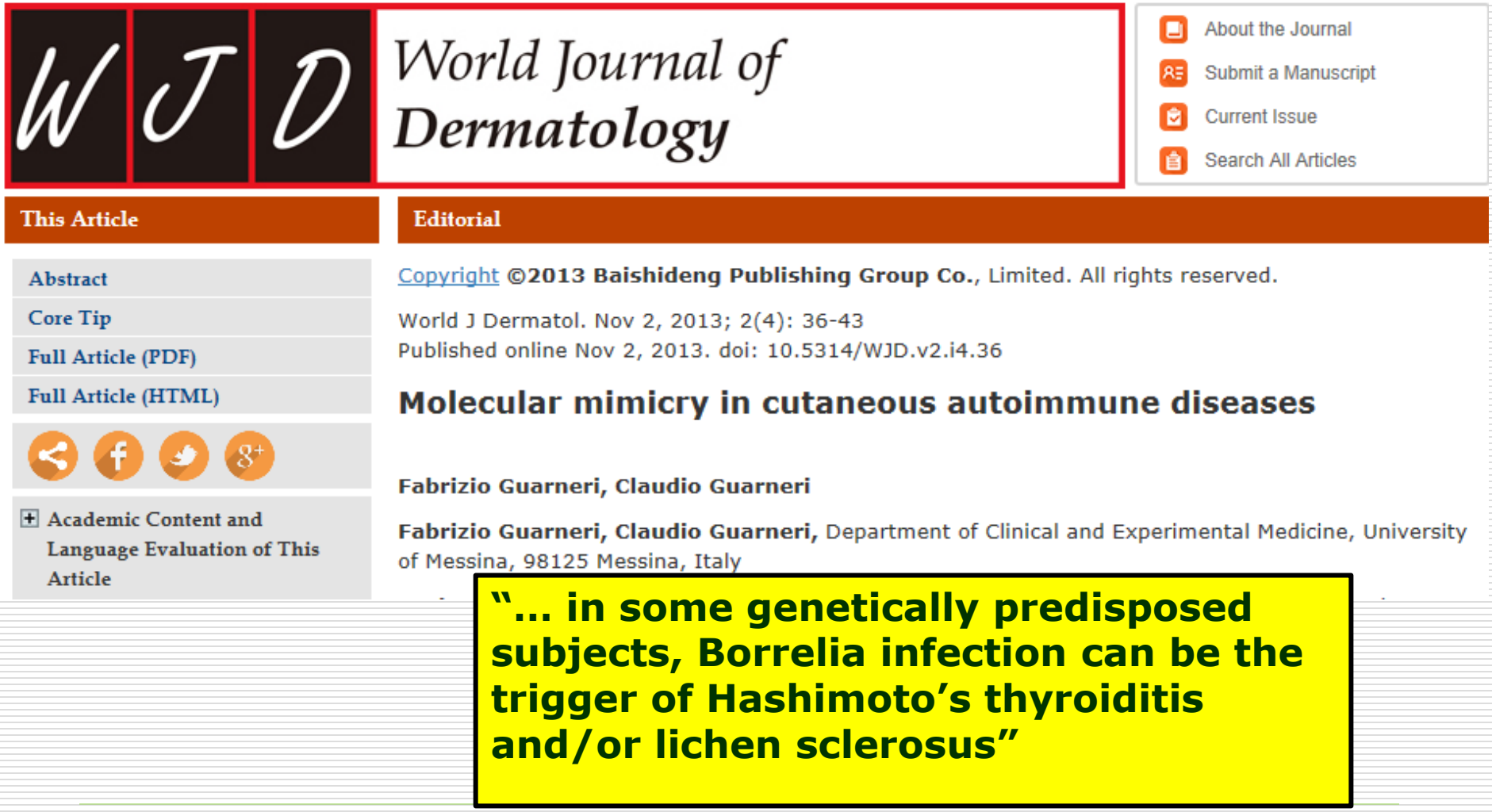
Abstract

Myalgic encephalomyelitis/chronic fatigue syndrome (ME/CFS) and fibromyalgia (FM) are chronic diseases with unclear aetiology. Human parvovirus B19 (B19) is immunomodulatory single-stranded DNA virus, which belongs to *Parvoviridae* family, *Parvovirinae* subfamily and *Erythrovirus* genus. B19 is considered as a possible pathogen or trigger factor in development of ME/CFS and FM.

Source: https://www.sciendo.com/article/10.2478/prolas-2019-0065;
https://www.rsu.lv/en/scientific-papers/human-parvovirus-b19-infection-status-patients-myalgic-encephalomyelitischronic

With autoimmunity, think pathogens too: Borrelia burgdorferi can trigger autoimmune thyroid disease (Hashimoto's)



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Editorial

World J Dermatol. Nov 2, 2013; 2(4): 36-43
Published online Nov 2, 2013. doi: 10.5314/WJD.v2.i4.36

Molecular mimicry in cutaneous autoimmune diseases

Fabrizio Guarneri, Claudio Guarneri

Fabrizio Guarneri, Claudio Guarneri, Department of Clinical and Experimental Medicine, University of Messina, 98125 Messina, Italy

"… in some genetically predisposed subjects, Borrelia infection can be the trigger of Hashimoto's thyroiditis and/or lichen sclerosus"

Borreliosis linked to Guillain-Barré Syndrome, Dermatomyositis and Sjögren`s Syndrome

Lyme disease symptoms can mimic many other illnesses and have been linked to several autoimmune diseases including Sjögren's syndrome [1], Dermatomyositis [2], and Guillain-Barre syndrome [3]. A case report by Smiyan entitled "Sjögren's syndrome and lymphadenopathy unraveling the diagnosis of Lyme disease," reflects the importance of a thorough clinical evaluation.

References:

1. Smiyan S, Galaychuk I, Zhulkevych I, et al. Sjogren's syndrome and lymphadenopathy unraveling the diagnosis of Lyme disease. Reumatologia. 2019;57(1):59-62.
2. Novitch M, Wahab A, Kakarala R, Mukerji R. The Emergence of a Forgotten Entity: Dermatomyositis-like Presentation of Lyme Disease in Rural Wisconsin. Cureus. 2018;10(5):e2608.
3. Patel K, Shah S, Subedi D. Clinical association: Lyme disease and Guillain-Barre syndrome. Am J Emerg Med. 2017.

Source: https://danielcameronmd.com/lyme-disease-manifests-as-autoimmune-disorder-sjogrens-syndrome/

Diseases caused by enteroviruses

PATHOLOGY

Table 4 Human diseases caused by enteroviruses

	Poliovirus	Coxsackie A virus	Coxsackie B virus	Echovirus	Enterovirus (other)
Asymptomatic infection	yes	yes	yes	yes	yes
Meningitis	yes	yes	yes	yes	yes
Paralysis	yes	yes	yes	yes	?*
Febrile exanthems	no	yes	yes	yes	yes
Acute respiratory disease	no	yes	yes	yes	yes
Myocarditis	no	yes	yes	yes	no
Orchitis	no	no	yes	yes	no

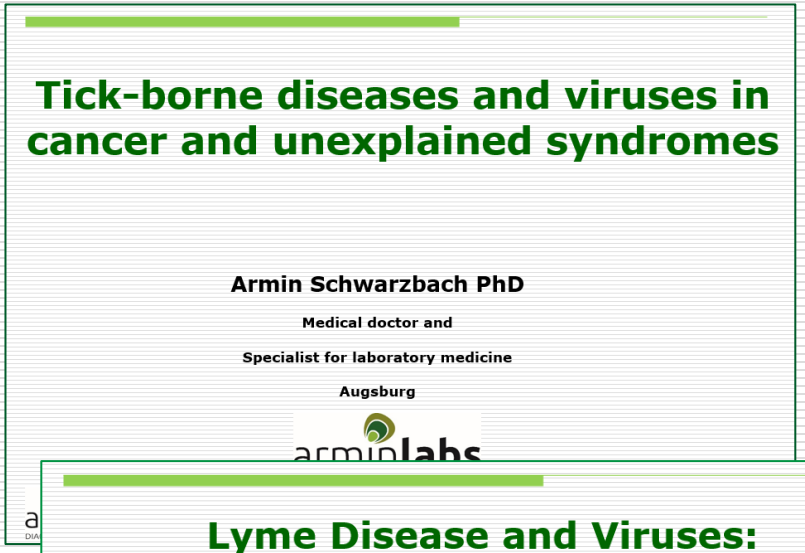
* Enterovirus-D68 (EV-D68) can replicate in blood and may damage the central nervous system. It has been detected in cerebrospinal fluid of patients with acute flaccid paralysis.

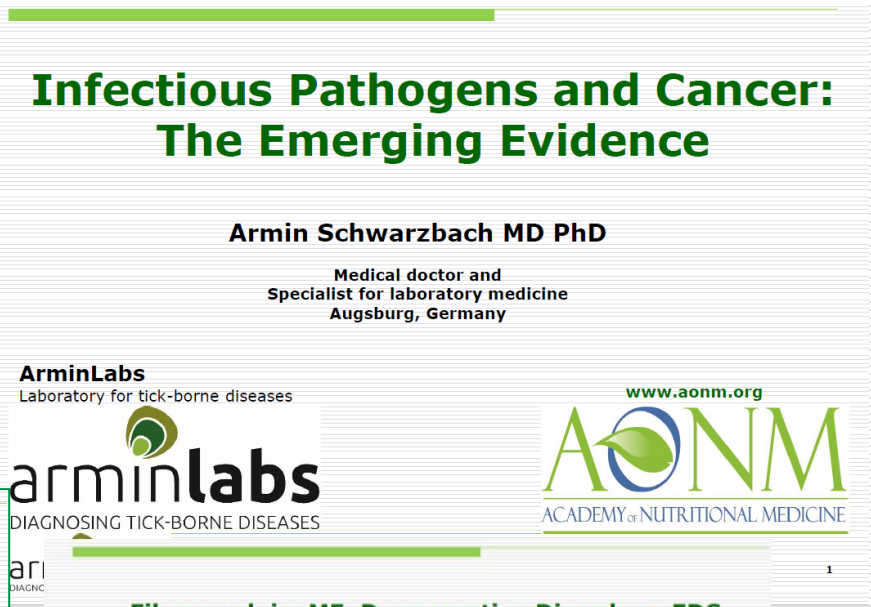
There have been reports of children hospitalized with muscle weakness or paralysis, usually in their arms and legs. They were tested for poliovirus, West Nile virus, and enteroviruses. About half of the children had EV-D68 in their nose secretions; usually, EV-D68 affects the respiratory system and it is not yet known if this respiratory infection is linked to their muscle weakness.

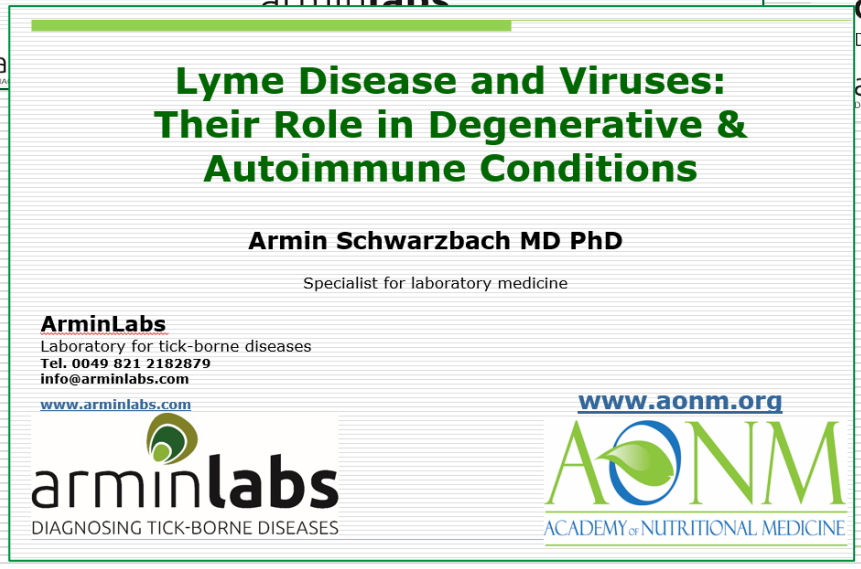
Source: http://www.microbiologybook.org/virol/picorna.htm

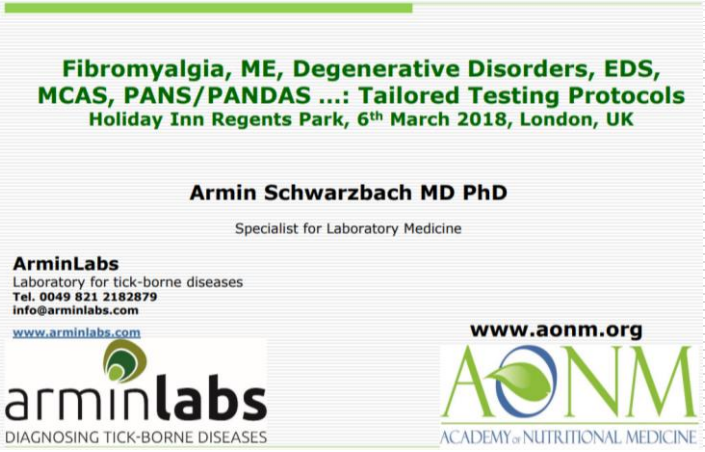
Lots of tailored testing protocols available …

https://aonm.org/viruses-and-testing/;
https://aonm.org/wp-content/uploads/2018/07/Tailored-Testing-Protocols-Mark-II-March-6th_V5_FINAL-2.pdf









… including panels specific to cancer (on request)

» Lymphoma/leukaemia/myeloma:

1. Borrelia ELISpot
2. Babesia ELISpot
3. Ehrlichia/Anaplasma ELISpot
4. Coxiella IgG/IgM antibodies
5. EBV ELISpot
6. CMV ELISpot
7. VZV ELISpot
8. CD3/ CD56/ CD57 cells

Monoclonal gammopathy:

1. Bartonella ELISpot
2. CD3/ CD56/ CD57 cells

Glioblastoma/brain cancer:

1. Borrelia-ELISpot
2. Mycolasma pneumoniae ELISpot + IgG/IgA antibodies
3. Toxoplasma IgG/IgM antibodies
4. CD3/ CD56/ CD57 cells

Prostate cancer:

1. Chlamydia pneumoniae ELISpot + IgG/IgA antibodies
2. Chlamydia trachomatis ELISpot + IgG/IgA antibodies
3. HSV1/2 ELISpot + IgG/IgA/IgM antibodies
4. CD3/ CD56/ CD57 cells

Lung cancer:

1. Chlamydia pneumoniae ELISpot + IgG/IgA antibodies
2. Mycoplasma pneumoniae ELISpot + IgG/IgA antibodies
3. CD3/ CD56/ CD57 cells

Angiogenic tumors:

1. Bartonella ELISpot
2. CD3/ CD56/ CD57 cells

Oesophageal cancer:

1. Mycoplasma pneumoniae ELISpot + IgG/IgA antibodies
2. CD3/ CD56/ CD57 cells

Breast cancer:

1. Bartonella Elisot
2. Mycoplasma pneumoniae ELISpot + IgG/IgA antibodies
3. CD3/ CD56/ CD57 cells

Cervical cancer:

1. Chlamydia trachomatis ELISpot + IgG/IgA antibodies
2. HSV1/2 ELISpot + IgG/IgA/IgM antibodies
3. CD3/ CD56/ CD57 cells

Many other tailored testing protocols available (examples)

Microbe-Disease correlations according to Armin Laboratories

arminlabs

» Cardiac Dysrrhythmias:

1. CMV ELISpot + IgG/IgM antibodies
2. EBV ELISpot + IgG/IgM antibodies
3. HSV 1/2 IgG/IgA/IgM antibodies
4. Coxsackie Virus IgG/IgA antibodies
5. Echovirus IgG/IgA antibodies
6. Rickettsia ELISpot
7. Borrelia ELISpot

» Uveitis:

1. Borrelia ELISpot
2. Tickplex basic
3. CD3/ CD56/ CD57 cells
4. Borrelia ELISpot
5. VZV ELISpot + IgG/IgA/ IgM antibodies
6. HSV1/2 ELISpot + IgG/ IgA/IgM antibodies
7. CMV ELISpot
8. Bartonella ELISpot
9. Chlamydia trachomatis ELISpot + IgG/IgA antibodies

» Skin rashes eg maculopapular exanthemata:

1. HHV-7ELISpot
2. Coxsackie Virus IgG/IgA antibodies
3. Echovirus IgG/IgA antibodies

» Rheumatoid Arthritis

1. Borrelia ELISpot
2. CD3/ CD56/ CD57 cells
3. Coxsackie Virus IgG/IgA antibodies
4. Echovirus IgG/IgA antibodies
5. Yersinia ELISpot
6. Campylobacter IgG/IgA antibodies
7. EBV ELISpot
8. CMV ELISpot
9. Mycoplasma pneumoniae IgG/ IgA antibodies
10. Chlamydia pneumoniae IgG/ IgA antibodies

Agenda

- Involvement of bacteria and viruses in multisystem disease
- **SARS-CoV-2/COVID-19 and viral reactivation**
- Testing
- Checklists and other resources

"Epstein-Barr virus (EBV) reactivation resulting from the inflammatory response to coronavirus infection may be the cause of previously unexplained long COVID"

pathogens

MDPI

Article

Investigation of Long COVID Prevalence and Its Relationship to Epstein-Barr Virus Reactivation

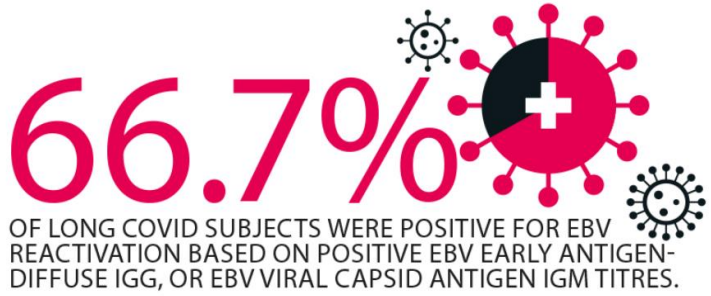
Jeffrey E. Gold [1,*], Ramazan A. Okyay [2], Warren E. Licht [3] and David J. Hurley [4]

[1] World Organization, Watkinsville, GA 30677, USA
[2] Department of Public Health, Kahramanmaraş Sütçü İmam University, Kahramanmaraş 46040, Turkey; razim01@gmail.com
[3] Warren Alpert Medical School of Brown University, Providence, RI 02903, USA; warren.licht@brownphysicians.org
[4] College of Veterinary Medicine, University of Georgia, Athens, GA 30602, USA; djhurley@uga.edu
* Correspondence: jeff_gold@world.org

Abstract: Coronavirus disease 2019 (COVID-19) patients sometimes experience long-term symptoms following resolution of acute disease, including fatigue, brain fog, and rashes. Collectively these have become known as long COVID. Our aim was to first determine long COVID prevalence in 185 randomly surveyed COVID-19 patients and, subsequently, to determine if there was an association between occurrence of long COVID symptoms and reactivation of Epstein–Barr virus (EBV) in 68 COVID-19 patients recruited from those surveyed. We found the prevalence of long COVID symptoms to be 30.3% (56/185), which included 4 initially asymptomatic COVID-19 patients who later developed long COVID symptoms. Next, we found that 66.7% (20/30) of long COVID subjects versus 10% (2/20) of control subjects in our primary study group were positive for EBV reactivation based on positive titers for EBV early antigen-diffuse (EA-D) IgG or EBV viral capsid antigen (VCA) IgM. The difference was significant ($p < 0.001$, Fisher's exact test). A similar ratio was observed in a secondary group of 1
may occur soon afte
long COVID sympto
COVID-19 inflamma

check for updates

Citation: Gold, J.E.; Okyay, R.A.; Licht, W.E.; Hurley, D.J. Investigation of Long COVID Prevalence and Its Relationship to Epstein-Barr Virus Reactivation. *Pathogens* **2021**, *10*, 763. https://doi.org/10.3390/



Lead study author Jeffrey E Gold said: "We ran EBV antibody tests on recovered COVID-19 patients, comparing EBV reactivation rates of those with long COVID symptoms to those without long COVID symptoms. The majority of those with long COVID symptoms were positive for EBV reactivation, yet only 10% of controls indicated reactivation.

… In a subset of 68 COVID-19 patients randomly selected from those surveyed, 66.7% of long COVID subjects versus 10% of controls were positive for EBV reactivation based on positive **EBV early antigen**-diffuse **IgG**, or **EBV viral capsid antigen IgM** titres."

Source: https://thebiomedicalscientist.net/news/long-covid-linked-epstein-barr-virus, https://www.mdpi.com/2076-0817/10/6/763

COVID-19 can potentially cause reactivation of VZV

COVID-19 can potentially cause reactivation of VZV and subsequently have an additive effect in neurologic complications

CASE

COVID-19 Associated With Concomitant Varicella Zoster Viral Encephalitis

Pavan Patel, DO, Anishee Undavia, MD, Rabia Choudry, MD, Yan Zhang, MD, and Aparna M. Prabhu, MD, MRCP

Correspondence
Dr. Patel
Patelpav@einstein.edu

Neurology: Clinical Practice April 2021 vol. 11 no. 2 e219-e221 doi:10.1212/CPJ.0000000000000902

Coronavirus disease 2019 (COVID-19) is a novel infectious disease caused by severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2). Patients can be asymptomatic or symptomatic with severity determined by age and comorbid conditions. Common early symptoms are fever, cough, dyspnea, myalgia, headache, and diarrhea. In addition to respiratory complications, other systems involved include genitourinary, gastrointestinal, and cardiac.[1]

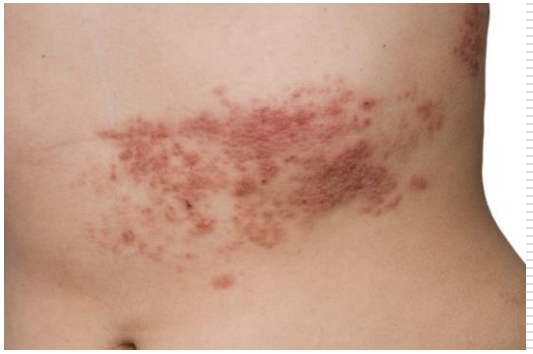
Neurologic complications such as encephalopathy were initially presumed to be because of multisystem involvement. Retrospective studies of patients with COVID-19 demonstrated multiple neurologic complications affecting central and peripheral nervous systems including dizziness, headache, hypogeusia, hyposmia, ischemic/hemorrhage stroke, and Guillain-Barre syndrome.[2] There was a single case report of hemorrhagic necrotizing encephalopathy reported in COVID-19, with imaging features of enhancement of bilateral thalami and medial temporal lobes.[3] To our knowledge, there have been no cases reported of coinfection with another virus during active COVID-19 infection resulting in neurologic manifestations.

PRACTICAL IMPLICATIONS

COVID-19 can potentially cause reactivation of VZV and subsequently have an additive effect in neurologic complications.

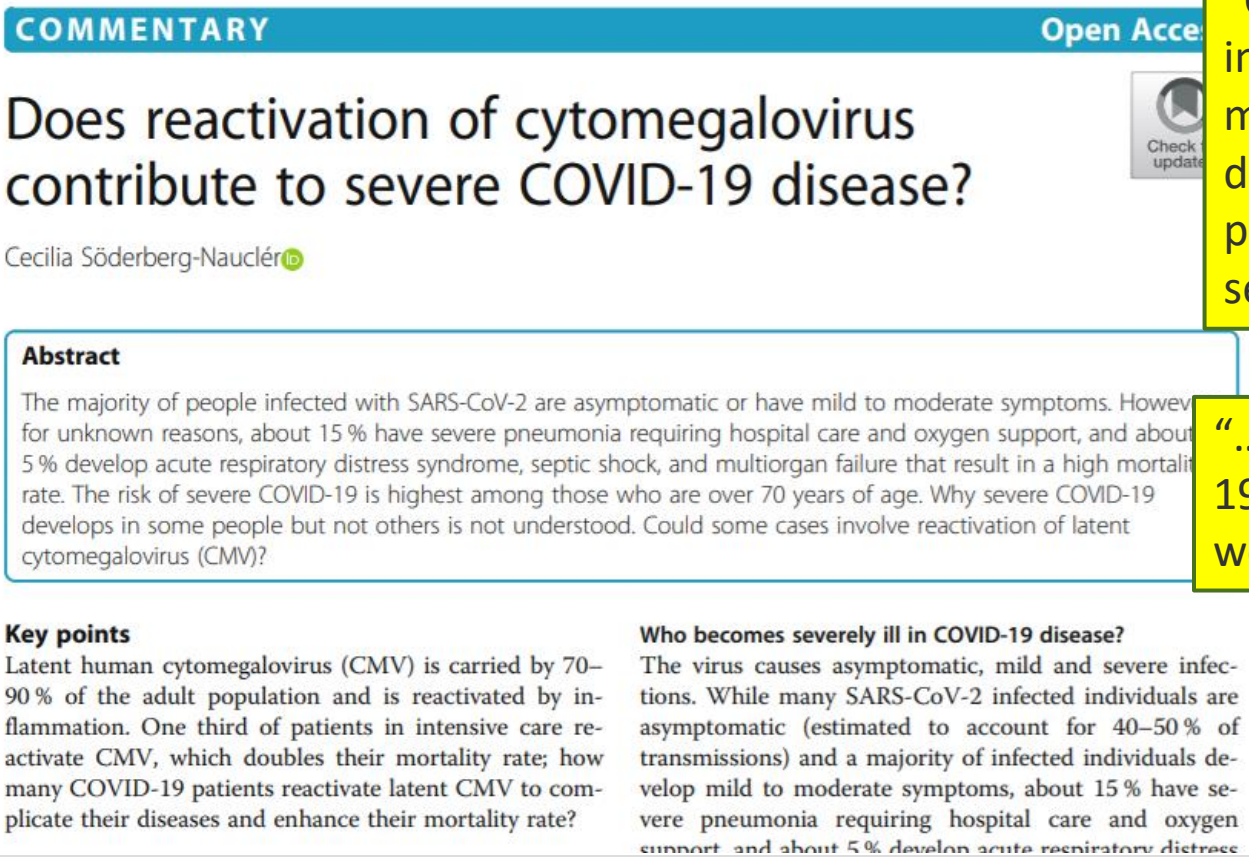
MORE ONLINE

COVID-19 Resources
For the latest articles, invited commentaries, and blogs from physicians around the world
NPub.org/COVID19



Source: https://cp.neurology.org/content/neurclinpract/11/2/e219.full.pdf; https://www.rheumatologyadvisor.com/home/general-rheumatology/herpes-zoster-reactivation-covid19-vaccination-autoimmune-inflammatory-rheumatic/

Cytomegalovirus and Covid-19



COMMENTARY Open Acce

Does reactivation of cytomegalovirus contribute to severe COVID-19 disease?

Cecilia Söderberg-Nauclér

Abstract

The majority of people infected with SARS-CoV-2 are asymptomatic or have mild to moderate symptoms. Howev for unknown reasons, about 15 % have severe pneumonia requiring hospital care and oxygen support, and about 5 % develop acute respiratory distress syndrome, septic shock, and multiorgan failure that result in a high mortali rate. The risk of severe COVID-19 is highest among those who are over 70 years of age. Why severe COVID-19 develops in some people but not others is not understood. Could some cases involve reactivation of latent cytomegalovirus (CMV)?

Key points

Latent human cytomegalovirus (CMV) is carried by 70–90 % of the adult population and is reactivated by inflammation. One third of patients in intensive care reactivate CMV, which doubles their mortality rate; how many COVID-19 patients reactivate latent CMV to complicate their diseases and enhance their mortality rate?

Who becomes severely ill in COVID-19 disease?

The virus causes asymptomatic, mild and severe infections. While many SARS-CoV-2 infected individuals are asymptomatic (estimated to account for 40–50 % of transmissions) and a majority of infected individuals develop mild to moderate symptoms, about 15 % have severe pneumonia requiring hospital care and oxygen support, and about 5 % develop acute respiratory distress

"CMV reactivation and virus induced immune dysfunction may be under-estimated as a driver of immuno-pathogenesis in patients with severe COVID-19."

"… diagnosing CMV in COVID-19 patients could be well worth the effort."

Source: *Söderberg-Nauclér, C. Does reactivation of cytomegalovirus contribute to severe COVID-19 disease?. Immun Ageing* ***18,*** *12 (2021). https://doi.org/10.1186/s12979-021-00218-z*

Herpes Simplex Virus reactivation with COVID-19

microorganisms

MDPI

Article

Herpes Simplex Virus Re-Activation in Patients with SARS-CoV-2 Pneumonia: A Prospective, Observational Study

Erica Franceschini [1,*], Alessandro Cozzi-Lepri [2], Antonella Santoro [1], Erica Bacca [3], Guido Lancellotti [3], Marianna Menozzi [1], William Gennari [4], Marianna Meschiari [1], Andrea Bedini [1], Gabriella Orlando [1], Cinzia Puzzolante [1], Margherita Digaetano [1], Jovana Milic [3], Mauro Codeluppi [5], Monica Pecorari [4], Federica Carli [1], Gianluca Cuomo [1], Gaetano Alfano [6], Luca Corradi [1], Roberto Tonelli [7], Nicola De Maria [8], Stefano Busani [9], Emanuela Biagioni [9], Irene Coloretti [9], Giovanni Guaraldi [3], Mario Sarti [4], Mario Luppi [10], Enrico Clini [7], Massimo Girardis [9], Inge C. Gyssens [11,12] and Cristina Mussini [1,3,*]

"Conclusions: our study shows a high incidence of HSV-1 re-activation both virologically and clinically in patients with SARS-CoV-2 severe pneumonia"

Our study has some strengths: it is the first study that analyzed the incidence and clinical implications of HSV-1 re-activation in patients with SARS-CoV-2 pneumonia; second it has strong clinical and therapeutic implications for COVID-19 patients, especially in the present and future waves of hospitalized patients most of whom are treated with steroids, which is now considered the SOC.

In conclusion, our study shows a high incidence of both virological and clinical HSV-1 re-activation in patients with SARS-CoV-2 severe/critical pneumonia. Data show an association between this risk and treatment with steroids, which could not be explained by age, previous IMV, and level of inflammation at hospital admission. Further studies are needed, especially a randomized controlled trial, to confirm the utility of acyclovir prophylaxis in COVID-19 patients with severe pneumonia admitted to the hospital.

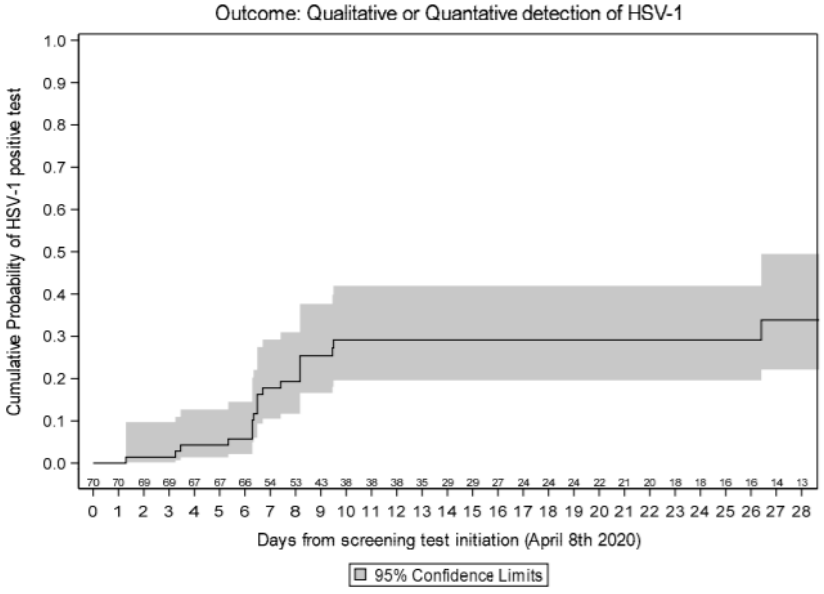


Figure 1. Kaplan–Meier estimates of HSV re-activation.

Source: https://www.msd-animal-health.ie/species/dogs/ticks/

Agenda

- Involvement of bacteria and viruses in multisystem disease
- SARS-CoV-2/COVID-19 and viral reactivation
- **Testing**
- Checklists and other resources

Testing can be either via antibodies, EliSpots, or PCR

The Humoral System: Antibody testing.
Antibody testing, often called serology, tests the B cell response. This consists of IgG (Immunoglobulin G), IgM (Immunoglobulin M), and (wherever possible) IgA (Immunoglobulin A).

The Cellular System: T-Cell immunity.
A technique for testing the other arm of the immune system, i.e., cellular T-cell immunity, is called EliSpot (enzyme-linked immunospot assay). This is a lymphocyte transformation test using an Interferon Gamma Release Assay.

PCR testing is available for all viruses, but is not as highly recommended in most cases as the two techniques above

IgM antibodies are the first type of antibody produced, and usually do not persist

IgM Antibody Functions and its Role in Disease

During infection, innate or "natural immunity" is provided by poly-reactive IgM antibody made by (B1a) B cells. IgM antibody acts to quickly recognize and initiate an immune response by directly neutralizing pathogens or clearing novel antigens. The three components of the IgM antibody-mediated immune response are activation of complement (C1qR and Fcα/μR), recruitment of phagocytic cells, and opsonization. Current research suggests that B1b B cells which make IgM antibodies may provide memory to certain pathogens and support T-cell independent immune responses. IgM antibody also acts as an educator of the immune system by transporting antigens to lymph tissues where memory is induced. Read more »

"Detection of IgM antibodies tends to indicate a recent initial exposure to an antigen, whereas detection of total or IgG antibodies indicates exposure some time ago."[2]

Source: 1. https://www.labtestsonline.org.au/learning/test-index/antibody-tests; 2. https://www.genscript.com/IgM-antibody.html

The difficulties of evidencing chronic (continuing) disease using IgG and IgM

In chronic disease, IgG may be there, but will be discounted as "past"; IgM will probably not be present

ENDOCRINOLOGY

Cytomegalovirus Ab(IgG)	183.0	AU/ml
	< 6.0 AU/mL is considered non-reactive	
	>=6.0 AU/mL is considered reactive	
Cytomegalovirus Ab(IgM)	Negative	
Comment	Result suggestive of previous CMV infection.	

IMMUNOLOGY

Epstein-Barr virus screen			
EBNA IgG antibody	* 36	U/ml	(< 5 U/ml Negative)
EBV Early Ag ab.(IgG)	<5	U/ml	(<10 U/ml Negative)
EBV VCA ab.(IgM)	<10	U/ml	(<20 U/ml Negative)
Comment	Results suggestive of past (latent) EBV infection.		

"IgG is produced in a delayed response to an infection and can be retained in the body for a long time …. IgG usually indicates a prior infection or vaccination."

Source: http://www.microbiologybook.org/mayer/Ab%20formation2000.htm

Some antibody tests show IgA status: this shows current activity along the mucosal membranes

Am J Kidney Dis. 1988 Nov;12(5):384-7.

The IgA mucosal immune system.

Lamm ME[1].

Author information

Abstract

This report reviews the immunophysiology of the mucosal immune system, the principal antibody of which is a special form of IgA, termed secretory IgA. This IgA is produced locally by mucosal plasma cells that are descended from precursors initially stimulated in organized, mucosal lymphoid organs designed for antigen sampling. After the initial triggering, the precursor cells pass via regional lymph nodes, lymph, and blood to disseminate widely among mucosal sites. After secretion from a local plasma cell, IgA binds to an epithelial cell surface receptor and the complex passes through the epithelial cell into the secretions where it serves as a nonphlogistic immunologic barrier to inhibit uptake of antigens. The product
dy,
such as the phlogistic Ig
s the
interrelatedness of the v
f
diseases including IgA n

PMID: 3055963

"IgA is quantitatively the most important of the immunoglobulins" [1] "The major antibodies found on mucous membranes are secretory IgA, which function primarily by binding microorganisms and thereby preventing their contact with the host tissues."[2]

Source: 1. Mucosal Immunity, Stephen P. James, in Encyclopedia of Immunology (Second Edition), 1998, https://www.sciencedirect.com/topics/neuroscience/immunoglobulin-a; 2. Hanson, L., Andersson, B., Carlsson, B. et al. Infection (1985) 13(Suppl 2): S166.

Testing the other arm of the immune system: T-cells

Using T-cells to show a cellular response against antigens is much more sensitive, and **indicates active infection (in contrast to antibodies, which can remain for months or years long after an infection is gone)**. EliSpot (enzyme-linked immunosorbent spot) technology has long been used in Germany to do exactly this: it quantifies T-cells that secrete signature proteins (such as a given cytokine) against a specific antigen. The Borrelia EliSpot evaluates the number of spot-forming units using a stimulation index (SI) based on IGRA (Interferon Gamma Release Assay).

Humana Press; 3rd ed. 2018 edition (14 July 2018)

The Elispot technique reflects the current T-cellular activity of bacteria and viruses

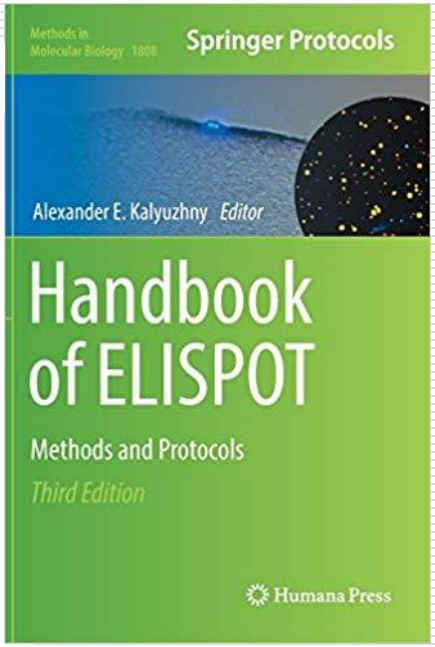
Chapter 1

Unique Strengths of ELISPOT for T Cell Diagnostics

Paul V. Lehmann and Wenji Zhang

Abstract

The T cell system plays an essential role in infections, allergic reactions, tumor and transplant rejection, as well as autoimmune diseases. It does so by the selective engagement of different antigen-specific effector cell lineages that differentially secrete cytokines and other effector molecules. These T cell subsets may or may not have cytolytic activity, can preferentially migrate to different tissues, and display variable capabilities to expand clonally. The quest of T cell immune diagnostics is to understand which specific effector function and T cell lineage is associated with a given clinical outcome, be it positive or adverse. No single assay can measure all of the relevant parameters. In this chapter, we review the unique contributions that ELISPOT assays can make toward understanding T cell-mediated immunity. ELISPOT assays have an unsurpassed sensitivity in detecting low frequency antigen-specific T cells that secrete effector molecules, including granzyme and perforin. They provide robust, highly reproducible data – even by first time users. Because
cytometry, ELISPOT is ideally su
ditions. These include defining (
establishing the fine–specificity
concentrations of the antigen in se
secretory products released by T
because T cells survive ELISPOT



"The quantification of single cell interferon-gamma (IFN-γ) release for assessing cellular immune responses using the Enzyme-linked immunospot (ELISPOT) assay is an invaluable technique in immunology."[1]

Source: 1 Sedegah M. The Ex Vivo IFN-γ Enzyme-Linked Immunospot (ELISpot) Assay Methods Mol Biol. 2015;1325:197-205; Humana Press; 3rd ed. 2018 edition (14 July 2018)

Example: Borrelia Elispot

Borrelia burgdorferi Elispot

Borrelia burgdorferi Full Antigen	+	32	SI
Borrelia b. OSP-Mix (OSPA/OSPC/DbpA)	+	29	SI
Borrelia burgdorferi LFA-1	(+)	2	SI

>3 = positive

2-3 = weak positive

<2 = negative

The results of the EliSpot-Tests indicate current cellular activity against Borrelia burgdorferi.

Key:
Immunodominant proteins: OSP = outer surface protein
DbpA = decorin-binding protein A
LFA = Lymphocyte Function Antigen 1
SI = stimulation index

```
  Borrelia EliSpot  *

1 Borrelia b. Full Antigen      !        15 SI
     0-1    = negative
     2-3    = weak positive
     > 3    = positive

1 Borrelia b. OSP-Mix           !        15 SI
     0-1    = negative
     2-3    = weak positive
     > 3    = positive

1 Borrelia burgdorferi LFA-1              0 SI
     0-1    = negative
     2-3    = weak positive
     > 3    = positive

     The results of the EliSpot tests indicate current cellular
     activities against Borrelia burgdorferi.
```

Example: Cytomegalovirus

CMV EliSpot

1 CMV Lytisch ! 22 SI
0-1 = negative
2-3 = weak positive
> 3 = positive

1 CMV Latent ! 2 SI
0-1 = negative
2-3 = weak positive
> 3 = positive

The result of the EliSpot test indicates current activity against Cytomegalo Virus (CMV).

Nov. 2021, before COVID diagnosis

CMV EliSpot

1 CMV lytic ! 355 SI
0-1 = negative
2-3 = weak positive
> 3 = positive

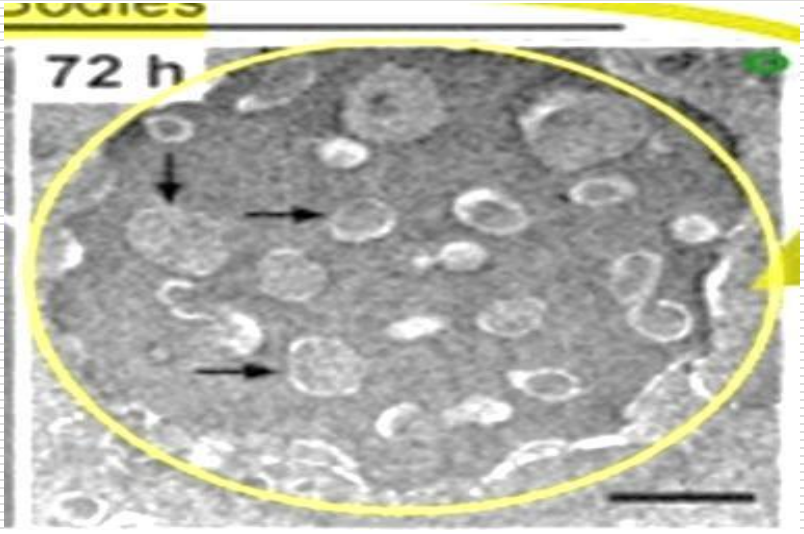
1 CMV Latent ! 106 SI
0-1 = negative
2-3 = weak positive
> 3 = positive

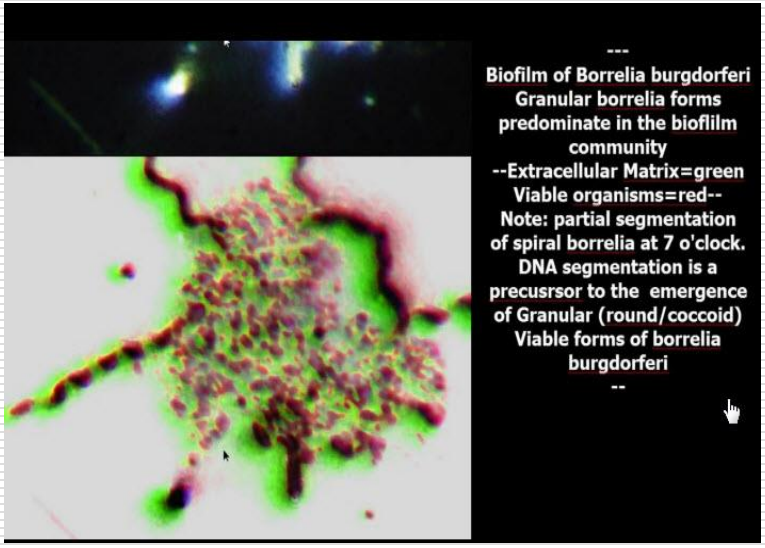
The result of the EliSpot test indicates current celluar activity against Cytomegalo Virus (CMV).

April 2022, after COVID

Source: ArminLabs results

Round bodies (pleomorphic forms) and biofilm-like colonies of Borrelia burgdorferi in vitro: Antibodies?

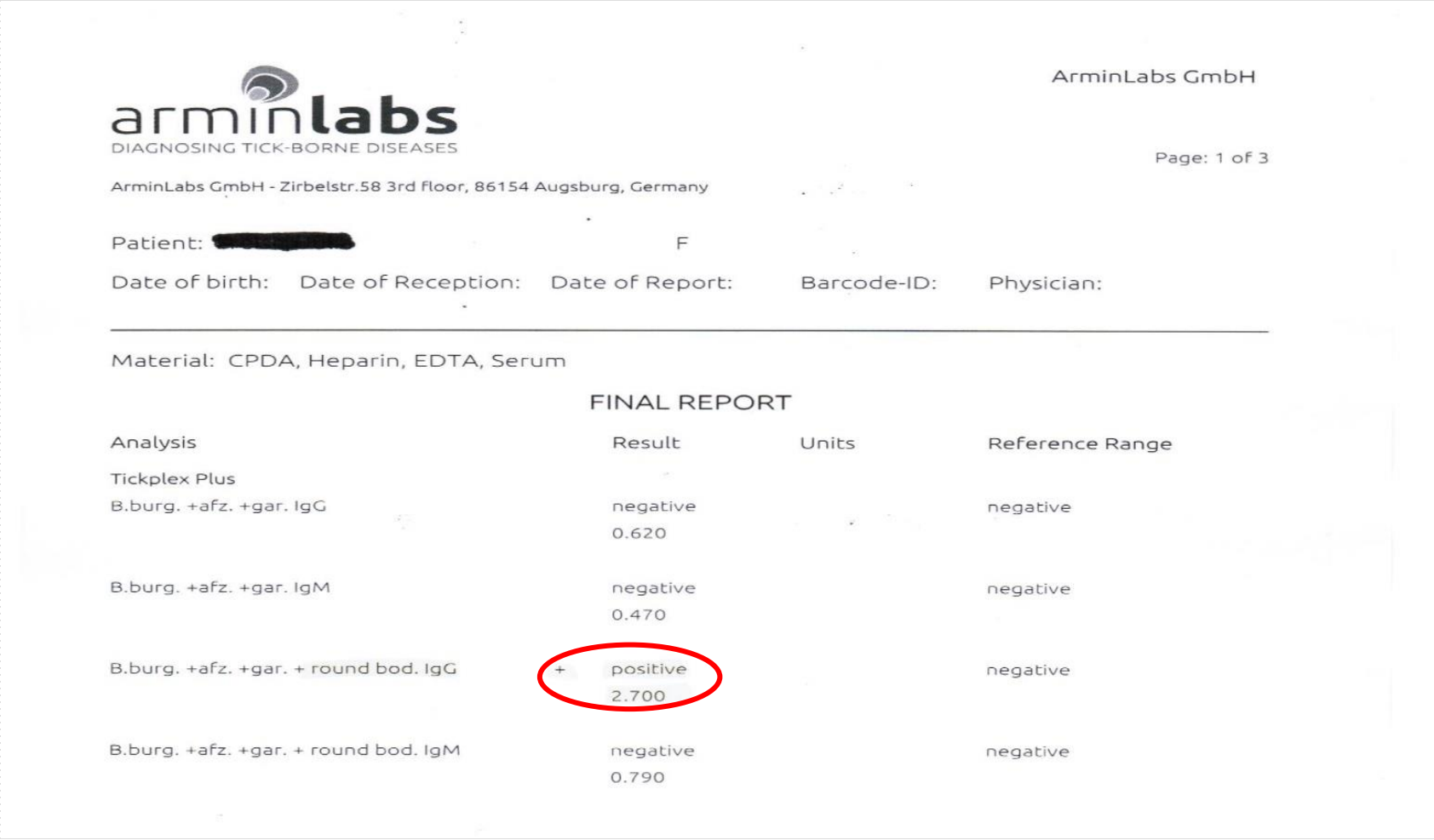




…pleomorphic B. burgdorferi should be taken into consideration as being clinically relevant and influence the development of novel diagnostics and treatment protocols…

Merilainen L., Herranen A., *Schwarzbach A.*, Gilbert L. Morphological and biochemical features of B.b. pleomorphic forms, Microbiology, published online ahead of print January 6, 2015, doi: 10/mic.0.000027

Tickplex: Highly sensitive multiplex methodology – only test in the world that can identify "round bodies"



arminlabs
DIAGNOSING TICK-BORNE DISEASES

ArminLabs GmbH

Page: 1 of 3

ArminLabs GmbH - Zirbelstr.58 3rd floor, 86154 Augsburg, Germany

Patient: [redacted] F

Date of birth: Date of Reception: Date of Report: Barcode-ID: Physician:

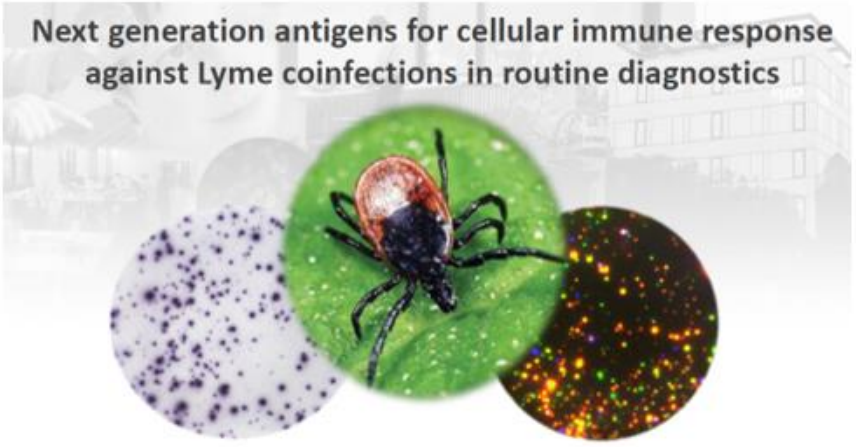
Material: CPDA, Heparin, EDTA, Serum

FINAL REPORT

Analysis		Result	Units	Reference Range
Tickplex Plus				
B.burg. +afz. +gar. IgG		negative 0.620		negative
B.burg. +afz. +gar. IgM		negative 0.470		negative
B.burg. +afz. +gar. + round bod. IgG	+	positive 2.700		negative
B.burg. +afz. +gar. + round bod. IgM		negative 0.790		negative

Elispot test for SARS-CoV-2 also available

3. A new Lyme test using the innate immune system: the Lyme iSpot



The **Lyme iSpot** provides information on the activity of a potential Borrelia burgdorferi infection. This test can differentiate between active (specific effector cells) and latent (specific memory cells) infections. Due to the **EliSpot (Enzyme-linked immunosorbent spot assay)**, it is now possible to better evaluate infection, inflammation and autoimmune processes.

While the **EliSpot** is exclusively based on interferon γ production, the **Lyme iSpot** also determines the cytokine IL-2. The IFN gamma and IL2 cytokine responses are measured using a "stimulation index" (SI). If the IFN gamma iSpot is positive (SI of 2-3 is weak positive, >3 is positive), this indicates that the sample contains effector T cells that are reactive to B. burgdorferi, and the patient is most likely to have an active infection. This suggests the advisability of therapy with antimicrobial protocols. If the IL2 iSpot is positive (same SI reference ranges), this indicates the presence of Lyme-specific memory cells. This indicates immune memory to past infection, but is also considered a "latent" signal. The decision as to whether to treat should be taken considering a patient's symptoms. The value of the iSpot is that it gives you two different perspectives on the reaction of the patient's immune system.

Checklists help decide which infections to test for; history and physical signs/symptoms also vital

arminlabs
DIAGNOSING TICK-BORNE DISEASES

AONM
ACADEMY of NUTRITIONAL MEDICINE

Short Symptom Checklist for Lyme Borreliosis

Name, first name ..... Date: .....

►	Actual and former symptoms: Please mark with a cross	X
1	Former or recent tick bite	
2	Former or recent bull´s eye rash	
3	Summer flu after tick bite	
4	Fatique/Malaise/Lethargy	
5	Loss of physical/mental capacity, general weakness	
6	Neck-pain, neck stiffness	
7	Headache	
8	Painful joints, swollen joints	
9	General aches and pains, tendon problems	
10	Muscle pain, muscle weakness	
11	Fever, feverish feeling, shivering	
12	Ears: intermittent red, swollen earlap	
13	Heart problems, disturbance of cardiac rhythm	
14	Cough, expectoration, breathlessness	
15	Night sweat	
16	Sleeplessness, waking up around ..... p.m.	
17	Tinnitus	
18	Swollen lymph nodes	
19	Numbness of the skin	
20	"Burning" or "pins and needles" skin sensations, painful sole or foot	
21	Back pain, back stiffness	
22	Occasional muscle twitching in the face, arms, legs	
23	Shivering, chill	
24	Blurred, foggy, cloudy, flickering, double vision	
25	Aggressiveness, drowsiness, panic attacks, anxiety, mood swings	
26	Concentration problems, short-term memory loss, forgetfulness	
27	Skin partly thin, paper-like, transparent, dry	
	Total number of symptoms for Lyme Borreliosis	

Antibiotics? When? Which one(s)? How long?

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DIAGNOSING TICK-BORNE DISEASES

ArminLabs GmbH
Zirbelstr. 58, 2nd floor
86154 Augsburg
GERMANY

Coinfections-Checklist

Name, first name ..... Date (DD/MM/YYYY) .....

	Actual and former symptoms Please mark with a cross	X	Score-Points (filled in by physician/naturopath)	Ranking
1	Stomach ache, gut problems		Ehrlichia&Anaplasma:	
2	Anaemia		Babesia:	
3	Diarhoea intermittent		Rickettsia:	
4	Fever or feverish feeling		Bartonella:	
5	Lack of concentration, memory disturbance, forgetfulness		Chl.pneumoniae:	
6	Encephalitis/Inflammation of the brain (NMR)		Chl.trachomatis:	
7	Yellowish colour of the skin/eyes		Yersinia:	
8	Painful joints, swollen joints		Mycoplasma:	
9	General aches and pains, tendon problems		Coxsackie-/Echo-Virus:	
10	Flu-like symptoms intermittent		EBV/CMV/HSV/VZV:	
11	Rash(es)			
12	Small red/purple spots of the skin			
13	Heart problems, disturbance of cardiac rhythm			
14	Cough, expectoration			
15	Headache			
16	Impaired liver function/ liver laboratory values			
17	Pneumonia, bronchitis			
18	Swollen lymph nodes			
19	Tonsilitis			
20	Enlargement of the spleen			
21	Fatigue / exhaustion, intermittent or chronic CFS			
22	Muscle pain, muscle weakness			
23	Shivering, chill			
24	Blurred, foggy, cloudy, flickering, double vision			
25	Nausea, vomiting			
26	Dark urine			
27	Itching or pain when urinating			

Electronic version fills automatically

Coinfections-Checklist

Name, first name ..... Date (DD/MM/YYYY) .....

	Actual and former symptoms Please mark with a cross	X	Score-Points (filled in by physician/naturopath)	Ranking
1	Stomach ache, gut problems	☒	Ehrlichia: 7	3
2	Anaemia	☐	Babesia: 4	6
3	Diarhoea intermittent	☐	Rickettsia: 6	4
4	Fever or feverish feeling	☒	Bartonella: 8	2
5	Lack of concentration, memory disturbance, forgetfulness	☒	Chl.pneumoniae: 9	1
6	Encephalitis/Inflammation of the brain (NMR)	☐	Chl.trachomatis: 5	5
7	Yellowish colour of the skin/eyes	☐	Yersinia: 5	5
8	Painful joints, swollen joints	☒	Mycoplasma: 7	3
9	General aches and pains, tendon problems	☒	Coxsackie-Virus: 9	1
10	Flu-like symptoms intermittent	☒	EBV/CMV/HSV: 7	3
11	Rash(es)	☒		
12	Small red/purple spots of the skin	☐		
13	Heart problems, disturbance of cardiac rhythm	☒		
14	Cough, expectoration	☐		
15	Headache	☒		
16	Impaired liver function/ liver laboratory values	☐		
17	Pneumonia, bronchitis	☐		
18	Swollen lymph nodes	☒		
19	Tonsilitis	☐		
20	Enlargement of the spleen	☐		
21	Fatigue / exhaustion, intermittent or chronic CFS	☒		
22	Muscle pain, muscle weakness	☒		
23	Shivering, chill	☐		
24	Blurred, foggy, cloudy, flickering, double vision	☒		
25	Nausea, vomiting	☒		
26	Dark urine	☐		
27	Itching or pain when urinating	☐		

Ranked in order of priority – draw for first place here: Chlamydia pneumoniae (CPN) and Coxsackie

Links to further information on testing on the website …

https://aonm.org/viruses-and-testing/

https://aonm.org/symptoms-list-of-lyme-disease-and-co-infections/

Which test for which virus?

British Society for Ecological Medicine: Spotlight on Chronic Infections

Hallam Centre, London, 8th February 2019

Armin Schwarzbach MD PhD
Medical Doctor and Specialist for Laboratory Medicine

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Symptom lists

Collatio

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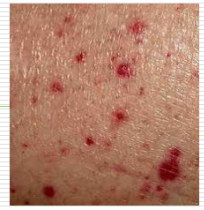
Clinical presentation of Babesia



- Headaches
- Night sweats
- Fevers
- Dry cough
- Air hunger
- Easy bruising
- Tinnitus
- Rage
- Despair
- Chills
- Flushing
- Sleep disturbance
- Vivid or violent dreams
- Dysphagia
- Psychic phenomena
- Severe neurological illnesses
- Thirst/Polydipsia
- Fatigue
- Rheumatoid arthritis
- Nausea (severe)
- Malaise
- Anaemia, thrombocytosis, thrombocytopenia
- Abdominal pain

How to obtain ArminLabs tests in the UK

- The Academy of Nutritional Medicine is the hub for ArminLabs tests in the UK. Please visit the ArminLabs tab on their webpage www.aonm.org
- The tab is:http://www.aonm.org/Armin%20Labs
- Their number is 03331 210 305
- They will advise you, arrange for a blood draw if requested, arrange a courier of the bloods to Germany, etc.

Thank you very much!

armin.schwarzbach@gmail.com

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